

ysis has abated. The Gd-MRI appearance tends to confirm the theory that Bell's palsy results from viral mononeuritis. Swelling of the nerve within the tight confines of the fallopian canal in the temporal bone results in ischemia and eventual neural dysfunction. The diameter of the nerve is the key difference between Bell's palsy and tumors involving the nerve. In Bell's palsy, the nerve is bright but not swollen or otherwise distorted, whereas in tumors, the nerve is typically both bright and focally enlarged. Gadolinium-MRI scans of patients with facial palsy should include the entire course of the facial nerve, from its exit from the brain stem to its terminal branches on the face.

In the recent past, clinicians managing a patient with atypical or persistent facial palsy faced a dilemma. If imaging studies were normal, the cause could be either atypical Bell's palsy or a subtle anatomic lesion beyond the resolving capacity of computed tomography or an unenhanced MRI study. The ability to make a positive diagnosis of Bell's palsy with Gd-MRI is reassuring to patients and clinicians. Indeed, many patients with Bell's palsy fear they have had a stroke, and an MRI scan may occasionally be warranted to allay those fears. A possible use for Gd-MRI in Bell's palsy stems from the preliminary observation that the degree of gadolinium enhancement correlates with the prognosis for the recovery of facial function. When intense gadolinium uptake involves a long segment of the nerve, a prolonged clinical course and incomplete recovery appear more probable.

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## Laser Therapy for Early Cancer of the Vocal Cords

"EARLY," SUPERFICIAL squamous cell cancer, limited to mucous membrane of the membranous portion of the vocal cord, can be treated with an 80% to 90% chance of cure with irradiation or surgical therapy. Surgical resection in the past was by cup forceps removal (stripping) of the mucosa of the membranous vocal cord. It is now done more precisely with a dry field using the laser through microdirect laryngoscopy.

For laser (or cup forceps) stripping to be successful, the cancer should not invade more than 1 to 2 mm below the basement membrane of the mucosa, and the best results, of course, are obtained when it is a squamous cell carcinoma in situ.

Normal vocal cord mobility is an indication that there is no invasion into Reinke's space just below the mucosa, much less into the thyroarytenoid muscle. This is confirmed with a suction tube tip by palpation of the involved vocal cord through direct laryngoscopy. The abnormal mucosa is then excised with a 1-mm normal mucosal margin and a thin layer of normal connective tissue under the lesion. The lesion needs to stop at least 1 mm from the anterior commissure, its

lateral edge should be visible on the floor of the ventricle (thus not go up into the false vocal cord), it should not extend onto the body and preferably not onto the vocal process of the arytenoid, and it should not go more than 3 to 4 mm below the free edge of the vocal cord. The margins should be clear and sharp.

The disadvantage of laser treatment is that irradiation achieves an equal five-year survival rate of about 90% and is equally effective when there is some invasion or spread slightly onto the arytenoid or onto the anterior opposite vocal cord or when margins are not sharply delineated. Although radiation therapy takes six weeks and can leave dryness in the throat, its main advantage is that it achieves a better voice on average than vocal cord stripping, even when the latter is done by an experienced laser surgeon through microdirect laryngoscopy. In summary, irradiation is advised as the primary therapy for T<sub>1</sub> and T<sub>2</sub>N<sub>0</sub>M<sub>0</sub> vocal cord squamous cell carcinoma. In carefully selected patients, however, especially after irradiation has failed, and if the lesion still meets (and originally met) the described criteria, laser resection of the lesion with a small margin around and beneath it may be a good alternative to vertical hemilaryngectomy or total laryngectomy.

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## Autoimmune or Immune-mediated Causes of Deafness

CERTAIN FORMS of rapidly progressive sensorineural hearing loss have an autoimmune or immune-mediated basis. Patients with these forms commonly have bilateral ear involvement that begins as fluctuating hearing loss but becomes progressive and often profound. There is an accompanying disequilibrium or outright ataxia if the vestibular system is also affected. About 20% to 30% have associated, definable manifestations of systemic autoimmune disease, such as systemic lupus erythematosus, polyarteritis nodosa, or ulcerative colitis. Diagnosis is difficult because no certain means exists to identify this disorder. The use of lymphocyte transformation against inner ear antigens has led to different results. Recent studies using a Western blot immunoassay against crude inner ear antigen have shown some diagnostic promise; of 54 patients with rapidly progressive hearing loss, 19 were positive on Western blot, but only 1 of the 14 (7%) normal persons showed a similar band.

Other means of evaluating this disorder include a sedimentation rate, the antinuclear antibody test, and C1q binding and Raji cell assays. These nonorgan-specific tests help identify a generalized autoimmune state in a patient.

Treatment consists of a trial of high-dose steroids—prednisone, 60 mg daily for three to four weeks. Improved hearing will often be evident after three weeks. Maintenance on alternate-day therapy should be attempted with as low a dose as possible, as long as hearing is maintained. Some investigators recommend giving cyclophosphamide—2 to 5 mg per kg of body weight a day—in patients who do not respond to steroids and who are not of childbearing age, as the drug has potential oncogenic risks.